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Assessment of Plantar Blood Flow and Carotid Intima Media Thickness in Type 2 Diabetic Peripheral Neuropathy Patients

HEBA M. IBRAHIM, M.D.; RASHA MOHAMED, M.Sc.; NEHAL HAMDY, M.D.;
NASHWA AL SARAF, M.D. and MAI G. ELSHENOUFY, M.D.

The Department of Internal Medicine, Faculty of Medicine, Cairo University

Abstract

Background: Diabetic peripheral neuropathy (DPN) causes disturbances in the foot blood flow, contributing to the etiology of diabetic foot ulcers and Charcot neuroarthropathy. Diabetic patients with microvascular complications appear to be at high risk of accelerated atherosclerosis.

Aim of Study: To assess the foot blood flow hemodynamic abnormalities, as well as carotid intima-media thickness (CIMT) measurement, which is eventual indicator of atherosclerosis that is not yet clinically apparent in type 2 DPN patients.

Patients and Methods: This observational cross-sectional study was carried outon sixty type 2 diabetic patients with DPN and sixty healthy controls. Using color Doppler ultrasonography, the volume flow rate and peak systolic velocity (PSV) in the plantar metatarsal arteries, posterior tibial artery, and dorsal is pedis artery were evaluated, as well as CIMT was measured.

Results: Compared to healthy controls, DPN patients have significantly higher volume flow rates and PSVs in the first, second, third, and fourth plantar metatarsal arteries as well as the lateral digital artery of the fifth toe (p-values ≤ 0.001). Regarding the volume flow rate and PSV in the dorsalis pedis and posterior tibial arteries, do not statistically differ between the two groups. DPN patients have a considerably thicker CIMT than healthy controls ($p<0.0001$).

Conclusions: DPN is linked to anobvious rise in the volume flow rate and PSV in the plantar metatarsal arteries. Moreover, DPN is highly associated with increased CIMT.

Key Words: Diabetic peripheral neuropathy – Blood flow – Plantar metatarsal arteries – Carotid intima-media thickness.

Correspondence to: Dr. Heba M. Ibrahim,
The Department of Internal Medicine, Faculty of Medicine,
Cairo University

Introduction

DIABETES is frequently described as a vascular disease, in which both microcirculation and macrocirculation are impacted, and the clinical consequences at the two vascular levels seem quite different **[1].**

DPNis a prevalent chronic microvascular complication that significantly impairs quality of life and leads to considerable disability **[2].** DPN includes distal symmetric polyneuropathy (DSPN), autonomic neuropathy, in addition to radiculo-plexopathies and mononeuropathies. DSPN is the most prevalent neuropathic syndrome among diabetics **[3].** Several pathophysiological mechanisms are involved in DPN development. Hyperglycemia and dyslipidemia are the key players. They contribute to mitochondrial dysfunction, inflammation, and oxidative stress, precipitating nerve dysfunction and cellular death. In addition, microvascular disease and impaired insulin signaling also contribute to DPN development **[4].**

It was suggested that DPN could alter the hemodynamics of blood flow in the small foot arteries, Moreover, Autonomic neuropathy leads to increased blood flow to the skin and bone **[5].** These disturbances in the foot blood flow may contribute to foot ulceration and Charcot neuroarthropathy. Therefore, diagnosing the extent of blood flow abnormalities in DPN patients' feet is of vital importance **[6].**

Moreover, diabetic patients with microvascular complications seem to be highly vulnerable to accelerated atherosclerosis. Atherosclerosis is the primary cause of cardiovascular disease (CVD) **[7].** Early detection of atherosclerosis is essential to pre-

dict and prevent CVD. Carotid intima-media thickness (CIMT) is a well-established and widely used marker of subclinical atherosclerosis and hence early detection of cardiovascular risk **[8]**.

Patients and Methods

Study design and participants:

The patients enrolled in this study were enlisted from the Diabetes and Endocrinology out patient clinic at Kasr Al-Ainy Hospital, Cairo University, between January 2023 and November 2023. This is a cross-sectional observational study that was performed on sixty patients with type 2 DM with DPN and sixty healthy controls. Each participant was subjected to comprehensive history taking and detailed medical history was obtained including age, gender, duration of diabetes and presence of other comorbidities. The neuropathic symptoms including pain, tingling and numbness were comprehensively assessed. Moreover, complete clinical examination including blood pressure measurement and careful examination of both feet. Body mass index (BMI) was calculated by dividing the weight in kilograms by height in square meters. The following clinical tests were done to assess peripheral neuropathy: – Pinprick sensation test. – 128-Hz tuning fork test. – Ankle reflex.

Laboratory tests:

HbA1c – fasting blood glucose – 2-hour postprandial blood glucose – Lipid profile including: Total cholesterol Triglycerides, LDL-C (Low-density lipoprotein cholesterol) HDL-C (High-density lipoprotein cholesterol) were done.

Imaging assessment:

Using a high-resolution color-coded Doppler ultrasound imaging system (Philips iU22 xMATRIX ultrasound system, USA) with a L9-3 linear transducer.

1- *The volume flow rate and peak systolic velocity (PSV):* In the plantar metatarsal arteries Fig. (1), posterior tibial artery, and dorsalis pedis artery were assessed, The mean value of each parameter in each artery was calculated by the sum of its readings from the right and left sides divided by two Fig. (2A,B).

The participants were examined while they were in the supine position. They were told to lie down and relax for ten minutes before performing the test. The room's temperature was kept at 25°C to ensure that temperature had no impact on the measurement outcomes. The volume flow rate was automatically measured by Doppler ultrasound. According to Gassner's analysis, the volume flow rate is equal to the cross-sectional area (A) × time-averaged velocity (TAV) **[9]**. Assuming that the vessel has a circular cross-section (such as arterial vessels), the A can be calculated as $\pi \times$ radius2 (or its equivalent, D2 × 0.785). The time-averaged peak velocity or TAP, across cardiac cyclescan be used to estimate TAV **[10]**.

2- *CIMT measurement:* CIMT is measured from the intima to the media of the carotid arterial wall. Our patients were assessedin the supine posture, with their necks extended anda slight head rotation in the direction opposing the examined carotid artery. The probe was placed in the anterolateral position. In the longitudinal section of the common carotid artery, the measurements were carried out at the point of maximum thickness on the posterior wall at 10mm before its bifurcation Fig. (3). The mean values from the left and right sides were added up and divided by two to determine the averaged CIMT. A CIMT of greater than 0.9mm is considered abnormal in accordance with the 2018 ESC/ESH guidelines for the treatment of arterial hypertension.

Statistical analysis:

All analyses were done using the Statistical Package of Social Science (SPSS, version 25). The mean, standard deviation, and lowest and maximum of the range were used to convey quantitative data, whereas frequency and percentage were used to express qualitative data. The independent sample *t*-test was used to compare means, while the Chi-square test was used to compare proportions. All statistical tests were two-tailed. The cut-off point for all significant tests was a probability of less than 0.05. Pearson correlation analysis was utilized to evaluate correlations among various variables.

Results

Demographic and laboratory parameters are illustrated in Table (1). Compared to healthy controls, DPN patients had a considerably higher BMI ($p<0.0001$). In addition, there were no statistically significant differences between the two groups regarding gender distribution, age, and the measurements of blood pressure.

Table (1) demonstrates that DPN patients had significantly greater levels of HbA1c, fasting blood glucose, 2-hour postprandial blood glucose, total cholesterol, LDL-C, triglycerides, and lower levels of HDL-C compared to healthy controls (all *p*-values <0.0001).

Imaging parameters are shown in Table (2). There was a significant rise in the volume flow rate in the first plantar metatarsal artery ($p=0.001$), second plantar metatarsal artery ($p=0.001$), third plantar metatarsal artery ($p<0.0001$), fourth plantar metatarsal artery ($p<0.0001$) and the lateral digital artery of the fifth toe ($p=0.001$) in DPN patients

compared to controls. Fig. (4). Additionally, DPN patients had significantly higher peak systolic velocities than healthy controls in the first, second, third, and fourth plantar metatarsal arteries as well as the lateral digital artery of the fifth toe (all *p*-values <0.0001). Furthermore, none of the two groups' volume flow rate and PSV of the dorsalis pedis and posterior tibial arteries differ statistically. Fig. (4).

Table (1): Demographic data and clinical characteristics of the studied groups.

	Group		*p*-value
	Case N=60	Control N=60	
Age (years):			
Mean ± SD	49.52±8.06	50.10±5.86	0.6
Range	36-65	40-63	
Sex:			
Male:			
Count	30	30	0.9
%	50.0%	50.0%	
Female:			
Count	30	30	
%	50.0%	50.0%	
Duration of DM (years):			
Mean ± SD	9.12±6.13	-	0.8
Systolic BP:			
Mean ± SD	107.33±12.33	107.67±11.10	0.2
Diastolic BP:			
Mean ± SD	67.17±8.04	68.67±7.47	<0.0001*
Weight (kg):			
Mean ± SD	81.11±15.63	61.23±4.30	0.09
Height (m):			
Mean ± SD	1.60±0.06	1.61±0.02	<0.0001*
BMI (kg/m^2):			
Mean ± SD	31.84±5.86	23.57±1.27	<0.0001*
Fasting blood glucose (mg/dL):			
Mean ± SD	182.83±88.47	79.33±7.51	<0.0001*
2h post-prandial blood glucose (mg/dL):			
Mean ± SD	267.55±105.91	111.40±13.07	<0.0001*
HbA1c:			
Mean ± SD	9.23±2.03	4.97±0.23	<0.0001*
Total cholesterol (mg/dL):			
Mean ± SD	206.42±36.13	167.93±13.64	<0.0001*
Triglycerides (mg/dL):			
Mean ± SD	144.23±50.16	104.87±25.53	<0.0001*
HDL cholesterol (mg/dL):			
Mean ± SD	42.98±10.81	53.08±4.52	<0.0001*
LDL cholesterol (mg/dL):			
Mean ± SD	129.08±30.57	98.12±12.63	

* Significant level of *p*-value is <0.05

* The *p*-value of the means was calculated using the independent sample *t*-test and chi-square test.

In addition, Table (2) shows that DPN patients' CIMT was significantly higher than that of healthy controls (p<0.0001).

As demonstrated in Table (3), a statistically significant positive correlations were addressed between the volume flow rates in the first, second, third, and fourth plantar metatarsal arteries, the lateral digital artery of the fifth toe and BMI, duration of DM, HbA1c, total cholesterol, triglycerides, LDL-C, as well as CIMT. In addition, statistical negative correlations were observed between the volume flow rates in the same arteries and HDL-C. Additionally, no statistically significant difference was seen between the volume flow rate of posterior tibial and dorsalis pedis and all demographic, laboratory parameters and CIMT.

Moreover, there are statistically significant positive correlations between the peak systolic velocities in the first, second, third, and fourth plantar metatarsal arteries and the lateral digital artery of the fifth toe and BMI, duration of DM, HbA1c, total cholesterol, LDL-C, and CIMT, as illustrated in Table (4).

Furthermore, PSV of the posterior tibial and dorsalis pedis did not show a statistical significant difference from all demographic, laboratory, and CIMT parameters, with the exception of a statistically significant positive correlation between PSV of the posterior tibial artery and HbA1c.

Finally, Table (5), Fig. (5) demonstrates a no table positive correlation between the volume flow rate and PSV in the dorsalis pedis artery, posterior tibial artery, the four plantar metatarsal arteries, and the lateral digital artery of the fifth toe.

Table (2): Comparison of the volume flow rates and peak systolic velocities in the DPN patients with those in healthy controls.

	Group		*p*-value
	Case N=60 Mean ± SD	Control N=60 Mean ± SD	
Dorsalis pedisartery:			
Volume Flowrate (cc/min)	23.10±14.64	23.25±13.82	0.9
Peak systolic velocity (cm/s)	72.01±16.30	71.05±15.20	0.6
Posterior tibial artery:			
Volume Flowrate (cc/min)	26.47±17.48	26.77±16.67	0.8
Peak systolic velocity (cm/s)	72.22±14.31	71.03±13.83	0.5
First plantar metatarsal artery:			
Volume Flowrate (cc/min)	19.59±10.57	15.89±6.88	0.001*
Peak systolic velocity (cm/s)	65.92±18.97	55.72±14.65	<0.0001*
Second plantar metatarsal artery:			
Volume Flowrate (cc/min)	21.58±16.09	15.10±9.09	0.001*
Peak systolic velocity (cm/s)	55.20±19.70	47.09±15.68	<0.0001*
Third plantar metatarsal artery:			
Volume Flowrate (cc/min)	18.39±12.51	12.56±7.31	<0.0001*
Peak systolic velocity (cm/s)	51.13±16.10	43.12±12.78	<0.0001*
Fourth plantar metatarsal artery:			
Volume Flowrate (cc/min)	17.89±11.18	12.38±6.68	<0.0001*
Peak systolic velocity (cm/s)	43.59±14.24	36.79± 11.17	<0.0001*
The lateral digital artery of the fifth toe:			
Volume Flowrate (cc/min)	14.02±11.67	9.69±8.92	0.001*
Peak systolic velocity (cm/s)	36.83±9.88	31.96±8.65	<0.0001*
CIMT	0.91±0.14	0.69±0.06	<0.0001*

* Significant level of p-value is <0.05

* The p-value of the means was calculated using the independent sample t-test.

Table (3): Correlations between the volume flow rates in the plantar metatarsal arteries and demographic data, clinical characteristics, laboratory investigations, and CIMT.

Pearson correlation	Volume flow of the first plantar metatarsal artery		Volume flow of thesecond plantar metatarsal artery		Volume flow of the third plantar metatarsal artery		Volume flow of the fourth plantar metatarsal artery		Volume flow of the lateral digital artery of the fifth toe		Volume flow rate in dorsalis pedis artery		Volume flow rate in posterior tibial artery	
	r	P-value	r	P-value	r	P-value	r	P-value	r	P-value	r	P-value	r	P-value
Age	0.09	0.141	0.06	0.295	0.003	0.966	0.004	0.946	0.03	0.558	0.001	0.988	0.049	0.453
Duration of DM	0.21	0.018*	0.11	0.041*	0.12	0.046*	0.19	0.031*	0.24	0.007*	0.155	0.091	0.181	0.428
BMI	0.25	0.019*	0.27	<0.0001*	0.26	<0.0001*	0.30	<0.0001*	0.27	0.007*	0.034	0.601	0.017	0.795
HbA1c	0.11	0.043*	0.15	0.019*	0.17	0.006*	0.19	0.002*	0.12	0.041*	0.049	0.449	0.046	0.477
Total cholesterol	0.20	0.027*	0.21	0.001*	0.20	0.002*	0.17	0.006*	0.19	0.013*	0.065	0.317	0.066	0.306
Triglycerides	0.30	0.001*	0.35	<0.0001*	0.29	<0.0001*	0.29	<0.000*	0.26	0.001*	0.112	0.085	0.036	0.581
HDL cholesterol	-0.17	0.006*	-0.16	0.012*	–0.19	0.002*	–0.18	0.005*	–0.15	0.017*	–0.019	0.772	–0.083	0.198
LDL cholesterol	0.13	0.033*	0.15	0.015*	0.11	0.043*	0.11	0.041*	0.13	0.045*	0.011	0.865	0.020	0.760
CIMT	0.19	0.002*	0.17	0.008*	0.17	0.006*	0.14	0.021*	0.13	0.041*	0.059	0.362	0.099	0.127

Table (4): Correlations between the peak systolic velocities in the plantar metatarsal arteries and demographic data, clinical characteristics, laboratory investigations, and CIMT.

Pearson correlation	Peak systolic velocity of the first plantar metatarsal artery		Peak systolic velocity of the second plantar metatarsal artery		Peak systolic velocity of the third plantar metatarsal artery		Peak systolic velocity of the fourth plantar metatarsal artery		Peak systolic velocity of the lateral digital artery of the fifth toe		Peak systolic velocity in dorsalis pedis artery		Peak systolic velocity in posterior tibial artery	
	r	P-value	r	P-value	r	P-value	r	P-value	r	P-value	r	P-value	r	P-value
Age	0.04	0.507	0.02	0.651	0.13	0.380	0.19	0.257	0.06	0.284	0.03	0.584	0.01	0.864
Duration of DM	0.17	0.041*	0.16	0.047*	0.19	0.042*	0.24	0.037*	0.20	0.047*	0.17	0.054	0.009	0.926
BMI	0.20	0.001*	0.24	<0.0001*	0.23	<0.0001*	0.15	0.014*	0.17	0.005*	0.12	0.051	0.01	0.863
HbA1c	0.21	0.001*	0.19	0.001*	0.22	<0.0001*	0.20	0.001*	0.24	<0.0001*	0.04	0.526	0.15	0.016*
Total cholesterol	0.22	<0.001*	0.13	0.041*	0.15	0.020*	0.12	0.048*	0.23	<0.0001*	0.02	0.683	0.06	0.340
Triglycerides	0.01	0.861	0.05	0.389	0.12	0.746	0.02	0.743	0.14	0.828	0.03	0.644	0.10	0.105
HDL cholesterol	-0.06	0.306	-0.02	0.657	-0.12	0.572	-0.03	0.629	–0.17	0.613	-0.02	0.698	–0.12	0.058
LDL cholesterol	0.19	0.003*	0.15	0.015*	0.12	0.041*	0.16	0.039*	0.17	0.018*	0.01	0.819	0.10	0.115
CIMT	0.26	<0.001*	0.19	0.003*	0.21	0.001*	0.28	<0.0001*	0.29	<0.0001*	0.04	0.535	0.09	0.152

Table (5): Correlations between the volume flow rates and peak systolic velocities.

Pearson correlation	The corresponding peak systolic velocity in the same artery	
	r	*p*-value
Volume flow rate in the dorsalis pedis artery	0.25	<0.0001*
Volume flow rate in the posterior tibial artery	0.43	<0.0001*
Volume flow rate in the first plantar metatarsal artery	0.41	<0.0001*
Volume flow rate in the second plantar metatarsal artery	0.15	0.014*
Volume flow rate in the third plantar metatarsal artery	0.19	0.002*
Volume flow rate in the fourth plantar metatarsal artery	0.16	0.012*
Volume flow rate in the lateral digital artery of the fifth toe	0.31	<0.0001*

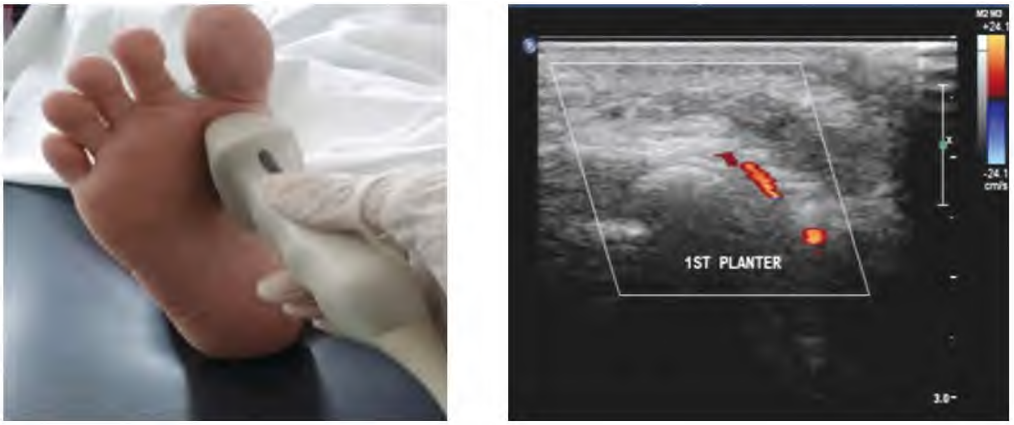


Fig. (1): Probelocation and Doppler ultrasound view of the first plantar metatarsal Artery.

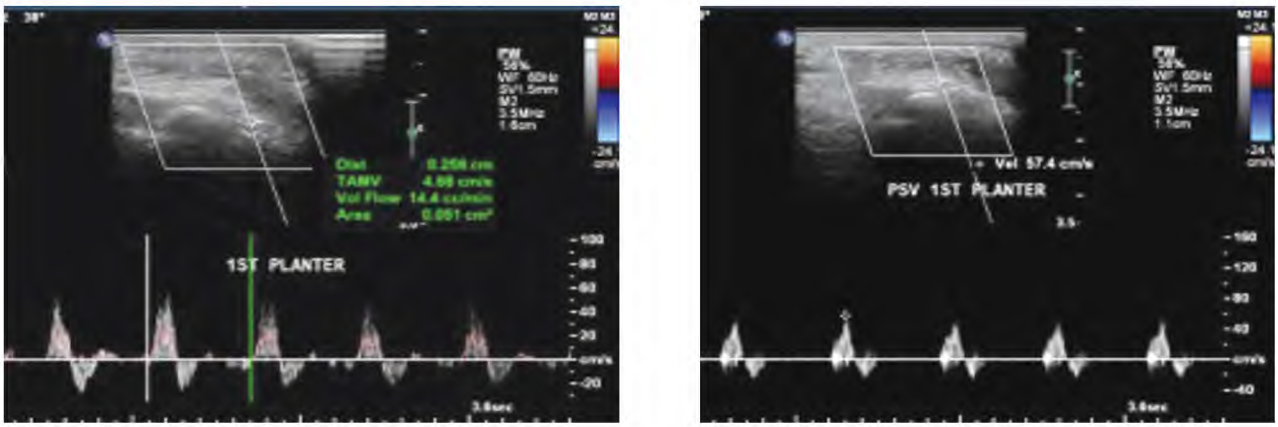
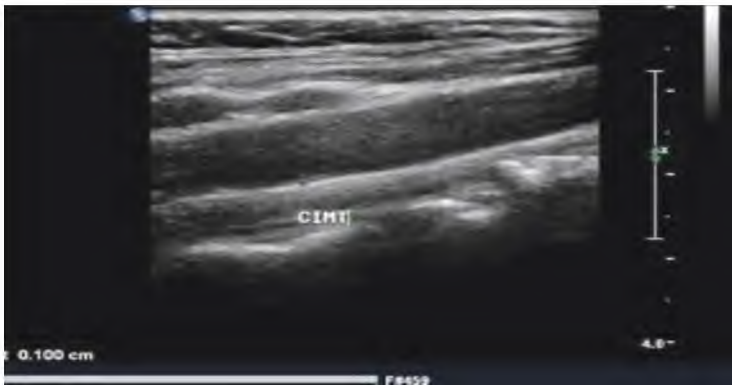


Fig. (2): (A): Doppler ultrasound view showing the volume flow rate in the first plantar metatarsal artery.
(B): Doppler ultrasound view showing PSV in the first plantar metatarsal artery.

Fig. (3): B mode of Doppler ultrasound determining CIMT.



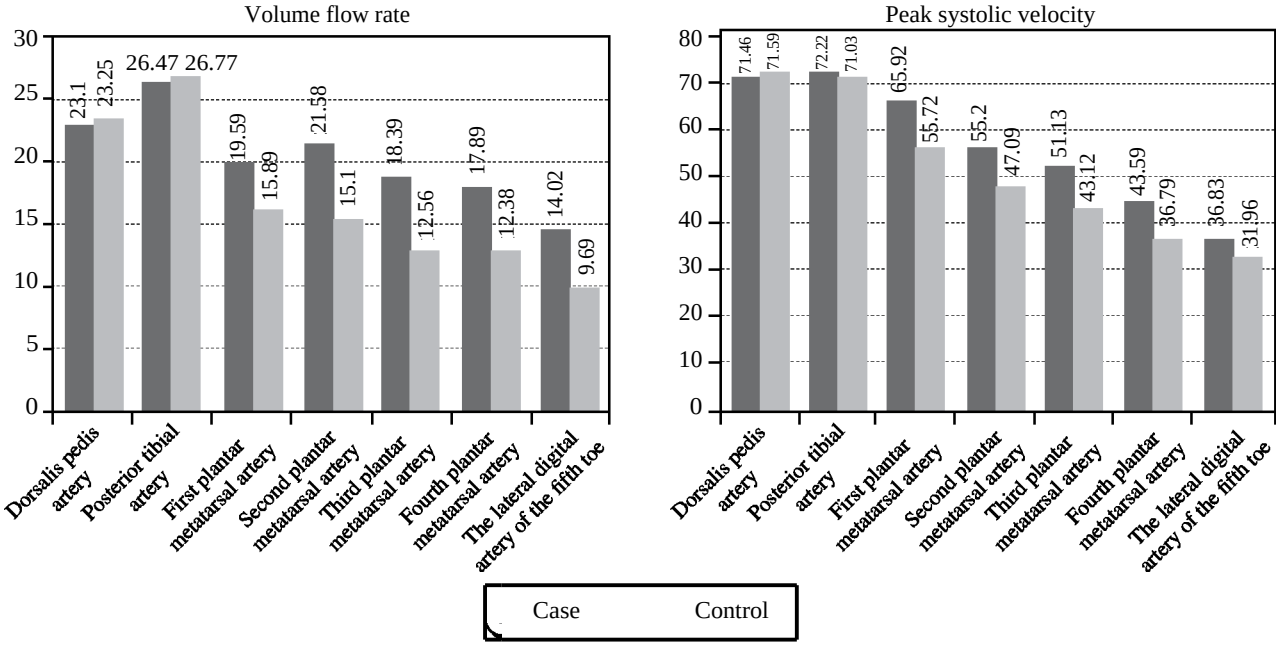


Fig. (4): Comparison of volume flow rates and peak systolic velocity in DPN patients with those in healthy controls.

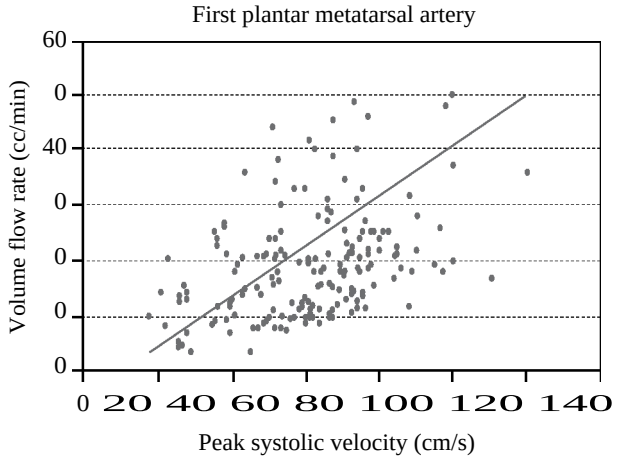


Fig. (5): Scatterplot showing the correlation between PSV and volume flow rate in the first plantar metatarsal artery.

Discussion

In the diabetic neuropathic foot with no evidence of occlusive arterial disease, DPN causes disturbances in the foot blood flow. Moreover, Jan et al., demonstrated that post-occlusive reactive hyperemia and poor thermal vasodilation are both compromised in individuals with DM and PN *[11]*. All of these are contributing factors to foot ulceration and Charcot neuroarthropathy *[12]*. According to previous researches, metabolic diseases and oxidative stress can lead to vascular alterations and damage to the blood vessels wall, which can impair peripheral nerve structure and function and result in DPN *[4,13]*.

It has been suggested that a strong interconnection between micro and macrovascular complications is present as microvascular diseases promote atherosclerosis *[14]*. Therefore, CIMT is used as a noninvasive indicator of subclinical atherosclerosis, which can predict the cardiovascular risk *[8]*.

Our results showed that BMI is noticeably greater in DPN patients than in healthy controls (p<0.0001). This is consistent with the findings of Jaid et al., *[15]* who observed that DPN patients (n=40) had a significantly greater BMI than healthy controls (n=40) (p=0.001). Furthermore, in line with our findings, Brown et al., in the UK showed that the DPN group (n=22) had a considerably higher BMI than the healthy control group (n=28) (p<0.01).

In addition, our study's findings demonstrated that DPN patients had significantly lower HDL-C and significantly higher total cholesterol, LDL-C, and triglycerides compared to healthy controls (all p-values <0.0001).

According to Jaid et al., *[15]* HDL-C was considerably lower and total cholesterol, LDL-C, and triglycerides were significantly higher in DPN patients (n=40) compared to healthy controls (n=40) (all p-values <0.05).

In addition, a study conducted by Vural and Gümüsyayla, *[16]* also reported that triglycerides levels were significantly higher (p=0.001), and HDL-C was significantly lower (p=0.02) in DPN patients (n=90) than in healthy controls (n=67). On the other hand, LDL-C and total cholesterol levels did not significantly differ between the two groups (p=0.29 and 0.47, respectively), which is discordant to our results.

Numerous studies have been conducted in the field of diabetic foot research to evaluate foot skin blood flow in DPN patients using laser Doppler flowmetry. Few studies, nevertheless, have examinedthe flow of blood in the foot's small arteries in those patients. Our study is among the few that assesses the volume flow rate and PSV in the plantar metatarsal arteries of people with DPN using Doppler ultrasound.

Our results showed a significant increase in the volume flow rate as well as the peak systolic velocities in all plantar metatarsal arteries in DPN patients in comparison with healthy controls. Furthermore, our results demonstrated that no statistically significant differences are present between DPN patients and healthy controls as regards the volume flow rate and PSV in the posterior tibial artery (p=0.8 and 0.5, respectively) as well as the volume flow rate and PSV in the dorsalis pedisartery (p=0.9 and 0.6, respectively).

This aligns with the findings by Zhou et al., *[17]* and Zhou et al., *[18]* whose research comprised 60 healthy controls and 60 type 2 diabetic patients with mild DPN. In the first, second, third, and fourth common plantar digital arteries as well as the fibular proper plantar digital artery of the first toe, both studies showed that the mild DPN group had significantly higher blood flow velocity and volume flow rate than the healthy control group (p<0.001). Furthermore, blood flow velocity and volume flow rate in the posterior tibial artery did not show anystatistical differences between the mild DPN group and the healthy control group.

It was suggested that the greater increase in blood flow was attributed to DPN patients' feet's arteriovenous shunts opening. These arteriovenous anastomoses are heavily innervated by sympathetic vasoconstrictor fibers *[19]*. In peripheral autonomic neuropathy, sympathetic denervation occurs, leading to their opening anddirection of the blood flow across these low-resistance, high-velocity, and high-volume arterial networks from the arteriolar side to the venular side *[20,21]*.

Moreover, Jan et al., *[22]* assessed the skin blood flow (SBF) in the DPN patients' feet using Laser Doppler flowmetry. Similar to our results, they reported that DPN patients (n=18) had significantly higher plantar and dorsal SBF than healthy controls (n=8; both p-values <0.05). Additionally, they noted that the plantar foot's SBF is considerably higher than the dorsal foot's in DPN patients (p<0.001).

In the present study, we assessed CIMT in type 2 DPN patients. Our results demonstrate that CIMTis considerably thicker in DPN patients than in healthy control participants (p<0.0001).

For instance, other studies have reported increased CIMT in DPN patients. Saluja et al., *[23]* whose study involved 200 type 2 diabetic patients, reported that CIMT was significantly higher among peripheral neuropathy subjects (n=112) than among diabetic patients without peripheral neuropathy (n=88) (p=0.0004) *[23]*. In addition, Guo et al., *[24]* performed a study using 247 type 2 diabetic patients, also demonstrated that CIMT in diabetic patients with dysfunctional small nerve fibers (n=144) was significantly greater than that in diabetic patients with normally functioning small nerve fibers (n=103) (p=0.018). Furthermore, Avci et al., *[25]* whose study included 161 type 2 diabetic patients, found that CIMT was significantly thicker in diabetic patients with DPN (n=69) than in diabetic patients without DPN (n=92) (p=0.01).

However, the study conducted by Kim et al., *[26]* recruited on 731 type 2 diabetic patients in South Korea, reported that no statistically significant difference was present between DPN patents (n=127) and patients who didn't have DPN (n=604) after adjusting for age and sex. The difference between studies may be due to ethnic variation.

Conclusion:

DPN is accompanied with a remarkable increase in the volume flow rate and PSV in small arteries of the foot (all plantar metatarsal arteries). As the changes that occur in the plantar metatarsal arteries blood flowmay be a reflection of the blood flow abnormalities in the regional microcirculation, which makes these patients may be greatly vulnerable to foot ulceration. In addition, we conclude that DPN is significantly associated with increased CIMT. DPN can be considered a determinant of subclinical atherosclerosis.

Recommendations:

Color Doppler ultrasonography is recommended as a noninvasive, easily performed, and widely available imaging technique to detect blood flow abnormalities in DPN patients' feet. Also, we recommend that those patients with increased blood flow strictly follow the instructions regarding foot care in addition to regular follow-up and regular foot examination for early detection of foot ulcers. Moreover, we recommend further prospective longitudinal cohort studies with the inclusion of a larger study population to track blood flow changes in the foot arteries and CIMT during DPN development and progression.

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تقييم تدفق الدم الخمصى وسماكة الوسط الداخلي للشريان السباتى فى مرضى السكرى من النوع الثانى المصابين بالاعتلال السكرى للاعصاب الطرفيه

يسبب الاعتلال العصبى المحيطى السكرى اضطرابات فى تدفق الدم فى القدم مما يساهم فى تقرح القدم واعتلال شاركو العصبى المفصلى. فى أقدام مرضى السكرى المصابين باعتلال الاعصاب المحيطية يحدث تحويل شريانى وريدى ايزداد تدفق الدم إلى الجلد والعظام.

يبدا أن مرضى السكرى الذين يعانون من مضاتفات الاتية الدموية الدقيقة معرضون بشكل كبير لخطر الاصابه بتصلب الشرايين المتسارع. مما يشكل خطر الرئيسى لمرضى القلب قياس سمك الطبقة الداخلية الوسطُى للشريان السباتى هو قياس غير جراحى يعامد تلى الموجات فوق الصوتية للشريان السباتى ويعتبر علامه على تصلب الشرايين تحت الكلينيكى وله علاقه بمخاطر القلب االاتية الدموية.

أجريت هذه الدراسه على مرضى المصابين بمرض السكرى من النوع الثانى مصابين باعتلال الاعصاب مقارنه باصحاء باستخدام التصوير بالموجات فوق الصوتية دوبلر تم تقييم معدل التدفق الحجمى وسرعه التدفق القصوى فى الشرايين المشطية الخمصية للشريان الخلفى والشريان القدم الظهرى كما تم قياس سمك الطبقة الداخلية الوطُى للشريان السباتى.

أظهرت النتائج أن معدل التدفق الحجمى وسرعه التدفق فى شراين مشط القدم الخمصى كانت أعلى بكثير مقارنة بالشخاص الاصحاء.

بالاضافة إلى ذلك، أظهرت النتائج أن سمُك الطبقة الداخلية الوسطى للشريان السباتى كان أعلى بكثير فى مرضى اعتلال الاعصاب المحيطى السكرى مقارنة بالاشخاص الاصحاء.